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Synchronized external pulsation is proposed as a method to improve tolerance to acceleration stress. This technique uses a modified anti-G suit which is pressurized and depressurized synchronously with the heart cycle. The feasibility of the procedure has been studied using a computer model of the cardiovascular system which includes the effects of Gz stress, and contains simulations of baroreceptor control of heart rate and venous tone. Model predictions indicate that for unprotected subjects, carotid pressure at eye level (ophthalmic artery pressure) decreases to 20 mmHg (beginning of central light loss) at approximately +3.6 Gz. Applying standard anti-G suit pressure to the model increases this level to 5.3 Gz. When synchronized external pulsation of 2 psi is superimposed on the standard anti-G suit pressure, the tolerance to acceleration stress is further augmented by at least 0.9 G above the protection afforded by the standard anti-G suit alone. A set of preliminary experiments on human subjects to test the feasibility of using the technique in the high G environment has also been carried out. The results under various protection

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SYNCHRONOUS EXTERNAL PULSATION FOR IMPROVED TOLERANCE TO ACCELERATION STRESS: MODEL STUDIES AND PRELIMINARY EXPERIMENTS

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SYNCHRONIZED EXTERNAL PULSATION FOR IMPROVED TOLERANCE TO ACCELERATION STRESS: MODEL STUDIES AND PRELIMINARY EXPERIMENTS

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ABSTRACT

Synchronized external pulsation is proposed as a method to improve tolerance to acceleration stress. This technique uses a modified anti-G suit which is pressurized and depressurized synchronously with the heart cycle. The feasibility of the procedure has been studied using a computer model of the cardiovascular system which includes the effects of G_z stress, and contains simulations of baroreceptor control of heart rate and venous tone. Model predictions indicate that for unprotected subjects, carotid pressure at eye level (ophthalmic artery pressure) decreases to 20 mmHg (beginning of central light loss) at approximately +3.6 G_z. Applying standard anti-G suit pressure to the model increases this level to 5.3 G_z. When synchronized external pulsation of 2 psi is superimposed on the standard anti-G suit pressure, the tolerance to acceleration stress is further augmented by at least 0.9 G above the protection afforded by the standard anti-G suit alone. A set of preliminary experiments on human subjects to test the feasibility of using the technique in the high G environment has also been carried out. The results under various protection modes compare favorably with the model predictions. Our results suggest that the computer model presented here is a useful tool for studying cardiovascular responses under $+G_z$ stress. It also indicates that using synchronized external pulsation pressure superimposed on the standard anti-G suit pressure may offer extra protection to acceleration stress.

INTRODUCTION

This report summarizes the results of a theoretical study whose purpose was to examine the possibility of using synchronized external pulsation (SEP) for augmenting tolerance to acceleration stress such as that developed by present day high performance aircraft. The study was carried out using a model of the cardiovascular system previously developed by us and adapted for acceleration studies [15,16].

Acceleration as it affects human subjects is usually described in terms of unit vectors G_x, G_y, and G_z, where the earth's gravitational acceleration is taken as a unit magnitude, and the z axis is the longitudinal axis of the body. Acceleration is most critical physiologically when it is in the +z direction. $+G_z$ acceleration causes a head to foot loading of the blood which can result in a reduction of flow to the brain. The first evidence of this reduction is a diminution of peripheral vision. Further increases in G_z produce loss of central vision, complete "black-out", and finally loss of consciousness.

The standard anti-G suit (AGS) is capable of increasing the tolerance to +z acceleration by 1 to 2 G [7,8]. The suit contains interconnected leg and abdominal bladders which are inflated to a pressure proportional to the +z direction acceleration experienced (pressure = 1.5 psi per G above 2 G). The pressure thus applied to the lower body can increase blood pressure in the central circulation, and prevent pooling of blood in the lower extremities. Certain muscular straining maneuvers (eg. M-1 or L-1 maneuvers) or positive pressure breathing (PPB) [27] which cause vascular compression and/or increased thoracic pressure can also be used to provide additional tolerance. These methods, however, still do not provide sufficient protection for pilots of today's high performance aircraft. In addition, the physical discomfort caused by the pressurized suit or by the mask used for PPB, and the severe fatigue caused by the M-1 or L-1 maneuvers make these methods less than ideal.

External pressure has also been used in the clinical setting to aid the function of the cardiovascular system. One device used for applying external pressure is the anti-shock trousers or counter pressure suit. This device is an inflatable pair of oversized trousers which can provide up to approximately 2 psi (apprximately 100 mmHg) of constant pressure to the abdomen and legs. Increases in arterial pressure of 15-35 mmHg through the use of this device have been reported in the literature [2,4,10,17,22,31]. The anti-shock trousers have also been operated in the so called 'phasic' mode by inflating the trousers to 45-50 mmHg for 45 seconds, followed by deflation to atmosphere for 15 seconds [13]. Using this technique, significant increases in right atrial pressure, systolic arterial pressure, and left atrial pressure have been reported.

The principle of the phasic mode has also been applied to the treatment of cardiac failure through the use of synchronized external pulsation (SEP), known clinically as external counterpulsation (ECP). Under this principle, pressure variations in the suit are synchronized to the cardiac cycle, with positive pressure applied during cardiac diastole. The suit is then deflated to atmosphere or to a negative pressure during cardiac systole. External pressure changes may be in the range of 200 to 250 mmHg. When

properly synchronized and phased, ECP can result in increases in cardiac output, diastolic pressure, and coronary flow, and in decreases in systolic pressure and cardiac oxygen consumption. ECP has been used as a non-invasive treatment for cardiogenic shock, and as early treatment of myocardial infarction [1,3,30,32]. The cardiovascular benefits afforded by the phasic mode of externally applied pressure suggest that this method may be a useful technique for augmenting G tolerance. One preliminary attempt to utilize this concept was previously reported [25]. The results were inconclusive due to equipment limitations.

While it is evident that externally applied pressure can provide definite G_z acceleration protection, the optimal form for this procedure is not clear. The investigation of methods to improve the effectiveness of external pressure can include animal experiments, human studies, or simulation. Animal studies are of limited value because of differences in blood volume distribution in the lower extremities of animals and humans. The use of human subjects for such studies precludes the direct measurement of many variables of interest. In either case, such experimental work is useful primarily for testing a particular procedure or system already devised. On the other hand, theoretical simulation can provide an insight into the interaction between the externally applied pressure and the cardiovascular system, leading to an optimal design of a G_z protection system.

We have developed a digital computer model of the human cardiovascular system which was used to study the effects of external pressure on the circulation and to assess new techniques for improved G_z tolerance. We have also begun a theoretical feasibility study of the use of SEP for increased G_z tolerance, and have performed preliminary experiments in human subjects to test the practical limitations of this method in a high G_z environment.

CARDIOVASCULAR MODEL

The basic model of the cardiovascular system, adapted for this study to predict the effects of externally applied pressure, has been reported in detail previously [15,16]. In brief, the model uses a variable compliance left ventricular simulation, a non-linear multielement arterial model consisting of 14 segments representing the aorta and 10 segments representing the carotid vessels and their branches, and lumped models of the systemic and pulmonary venous systems. As implemented on a digital computer, the model solves difference equations, based on the Navier-Stokes equations for fluid flow. To solve the equations, certain simplifying assumptions must be made. The most important are: blood is an incompressible Newtonian fluid; flow is one dimensional; velocity profile is flat except near the vessel wall; wall stress-strain relationships in the physiologic range are linear; arterial wall thickness is small compared to the vessel diameter; wall motion in the radial direction is proportional to the instantaneous volume stored in the segment; terminal arterial pressure-flow relationships are linear at low G and their behavior at high G may be represented as piecewise linear; during acceleration stress, cerebrospinal fluid pressure is equal to venous hydrostatic pressure at corresponding anatomical levels, thus preventing collapse of the venous channels within the skull [26]. The resulting equations for the nth segment of the arterial model are:

$$P_n = \frac{1}{C_n}\int (Q_n - Q_{n+1})\,dt + R_{2n}(Q_n - Q_{n+1})$$

$$Q_n = \frac{1}{L_n}\int (P_{n-1} - P_n - R_{1n}Q_n + P_{Gn})\,dt$$

$$P_{Gn} = \rho G_z l_n \cos\theta_n$$

$$r_n^2 = \frac{1}{\pi l_n}\int (Q_n - Q_{n+1})\,dt$$

where: $R = 8l\mu l_n/9\pi r_n^4$; $L = 9\rho l_n/4\pi r_n^2$; $C = 3\pi r_n^3 l_n/2Y_n h_n$; and $R_{2n} = 0.002/C_n$. P =blood pressure in the segment; Q_n=blood flow into the segment; P_{Gn}=hydrostatic effect of +z direction acceleration on the segment; r_n=radius of the segment; l_n =length of the segment; μ =blood viscosity; ρ =blood density; Y_n=Young's modulus of the segment; h_n=wall thickness of the segment; θ_n =the angle between the +z direction and the axis of the segment.

The effect of acceleration was incorporated into the model by the inclusion of a vectorial component of G_z as a hydrostatic pressure source in each model segment. Changes in the volumes of blood stored in each of the arterial and venous sections of the model at each end diastole were used to calculate left ventricular end diastolic volume. This volume was then used as input to the left ventricular model at the start of systole.

To use the model with externally applied pressure, additional assumptions and refinements were needed. These involved the coupling of external pressure to the vascular system, and the behavior of the vessels under changing transmural pressure. The external suit pressure was assumed to act on the lower arterial segments and on their peripheral branches (representing the abdomen and legs). Based on theoretical work on collapsible tubes by Kresch [19,20], we assumed that the behavior of the arterial branches can be characterized by two different compliance values. When, as a result of externally applied pressure, the vessel transmural pressure decreases to a value less than a threshold value, Po, the compliance (C) of the vessel increases substantially. Po is determined by Young's modulus, by the radius of the vessel, and by the thickness of the vessel wall. Po was estimated to be -10 mmHg for the abdominal aorta and the femoral artery, and -20 mmHg for the small arteries. At transmural pressures lower than Po, the compliances of the abdominal aorta and of the femoral artery were assumed to increase by a factor of 10, and that of the small arteries by a factor of 5.

The major branches of the arterial model terminate in lumped representations of arterial bed compliance and resistance, and venous pressure, Pv [16]. Coupling of the suit pressure to this peripheral vasculature was implemented by applying the pressure through the compliance element of the peripheral bed. Flow through the peripheral bed was assumed to cease below a critical closing pressure of 20 mmHg [5,9]. The direct effect of external pressure on the venous system has not been incorporated into the model as yet because, at present, this section of the simulation is represented by a lumped model.

The model contains simulations of the body's heart rate and venous tone control systems. Both are functions of pressure changes at the carotid section of the arterial model, and in turn, influence the operating state of the system [16]. Values of all parameters used in the model, and in the algorithms used for the heart rate and venous tone control systems were

taken from the literature [11,12,18,29].

SIMULATION PROCEDURES AND RESULTS

Acceleration stress was applied to the model at a 1 G/sec onset rate until the desired plateau level was reached. This level was maintained for 15 seconds. G_z was then reduced at a rate of 1 G/sec. This regimen corresponds to our experimental procedure described in the following section. Maximum simulated acceleration ranged from 2 to 6.5 G_z. Since visual impairment is a critical effect of high G_z stress on pilots, it was our goal to relate cardiovascular performance and light loss. There is at present, insufficient experimental information concerning the relationship between ophthalmic artery pressure and visual impairment. While some investigators have related peripheral and central light loss to systolic pressure levels [6,21], others have reported correlation with diastolic blood pressure levels [24,27]. For the purpose of the present study, we chose the criterion of Lambert and Wood [21]. In all cases of model simulation, central light loss (CLL) was assumed to occur at the G_z level for which systolic pressure in the ophthalmic artery decreases to 20 mmHg at any time during the run [6,21]. The start of peripheral light loss was defined as that G_z level at which systolic pressure drops below 50 mmHg.

The model was used to predict the effect on the circulation of several modes of external pressure protection during G_z stress. The results were compared to those obtained using no protection. The external pressure modes tested included: 1) standard AGS pressure applied to the legs and abdomen; 2) SEP applied only to the legs both with and without inflation of the abdominal segment of the AGS. (This mode was selected for simulation so that comparison with our preliminary experiments could be made); and 3) synchronized external pulsation superimposed on the standard AGS pressure. The external pressure was phased to provide augmentation during ventricular systole in order to increase vascular systolic pressure. Inflation was initiated at the beginning of systole and deflation was initiated at end systole. We tested a fast (50 msec) and a slow (125 msec) rate of inflation/deflation. The slow rate was chosen to permit comparison with our experimental results.

Typical results from the model simulations are shown in Figures 1 through 3. In these figures, a 4 G_z maximum acceleration profile was used. Figure 1 depicts the model's predictions for the unprotected subject. Figure 2 includes protection provided by the standard AGS, and Figure 3 illustrates the effects of 2psi of SEP pressure superimposed on the standard AGS pressure, with the SEP pressure applied during cardiac systole. Each figure depicts aortic root pressure, ophthalmic artery pressure, carotid flow, suit pressure, and the applied G_z profile. In those figures demonstrating results of runs with applied external pressure, suit bladder pressure is also included. The curves of Figures 1 through 3 are typical of most G_z levels tested, except for degree of response. One exception was that at G_z levels above 5, further decline of eye level pressure began toward the end of the 15 second plateau.

Table 1 summarizes the model's predictions regarding peripheral light loss (PLL) and central light loss (CLL) for each protection mode tested.

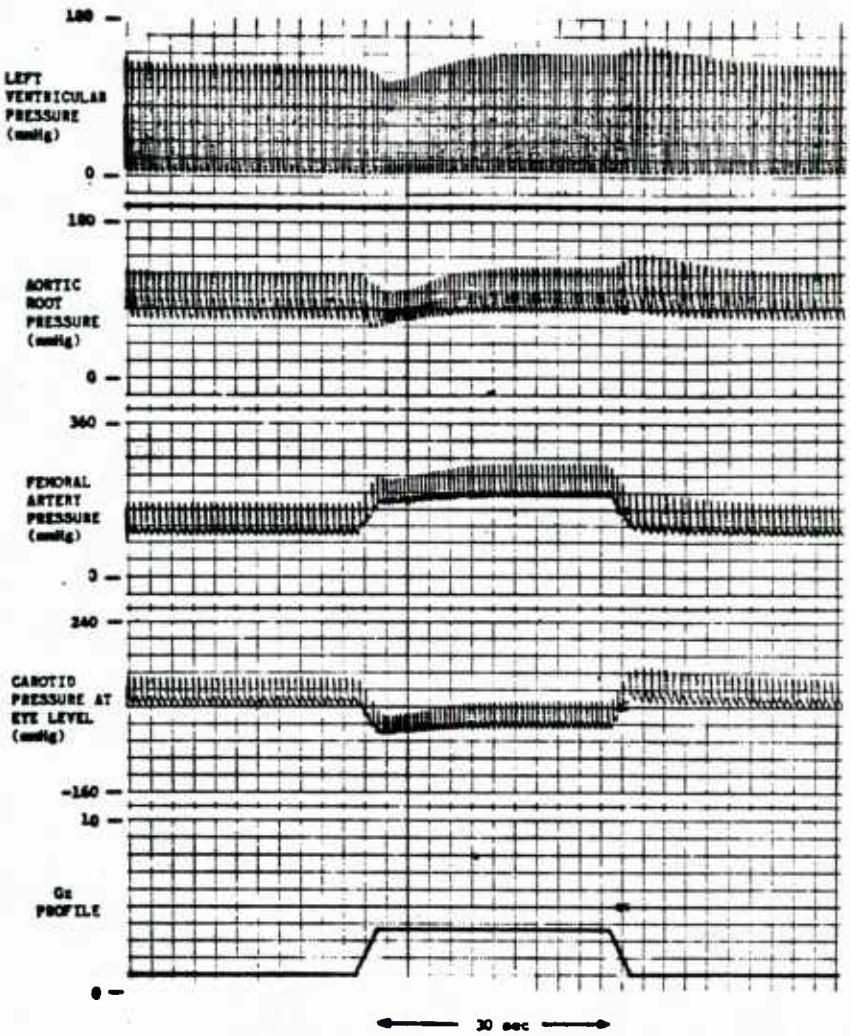

Figure 1 - Model Output for Unprotected Subject. See text for details.

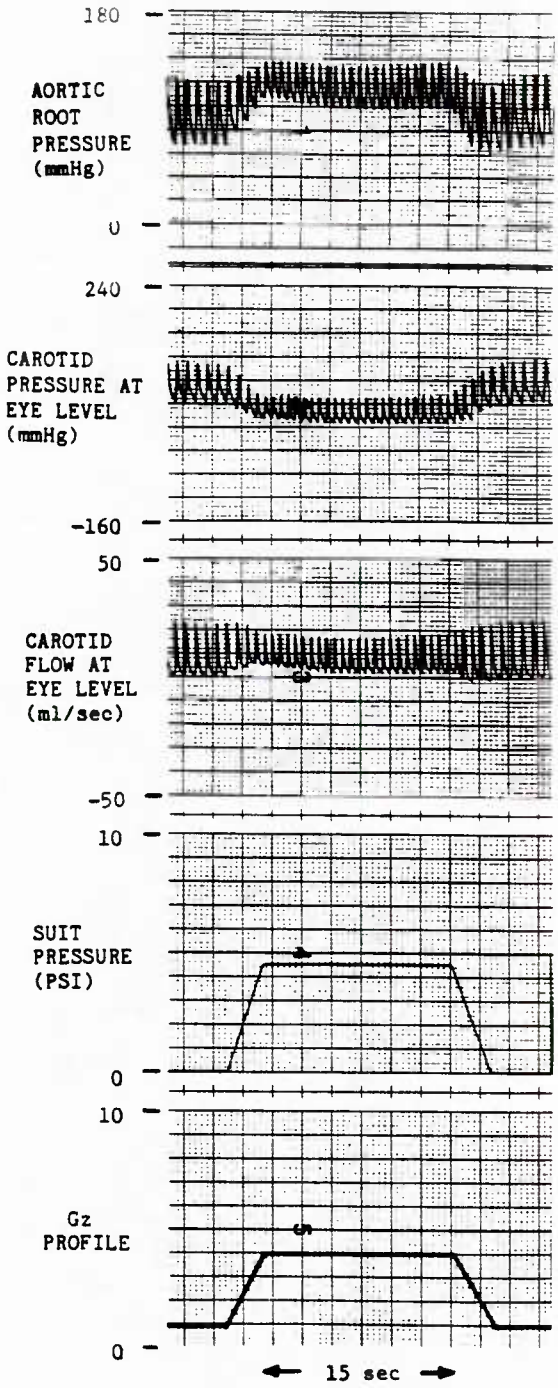

Figure 2 - Model output for subject wearing standard anti-G suit. See text for details.

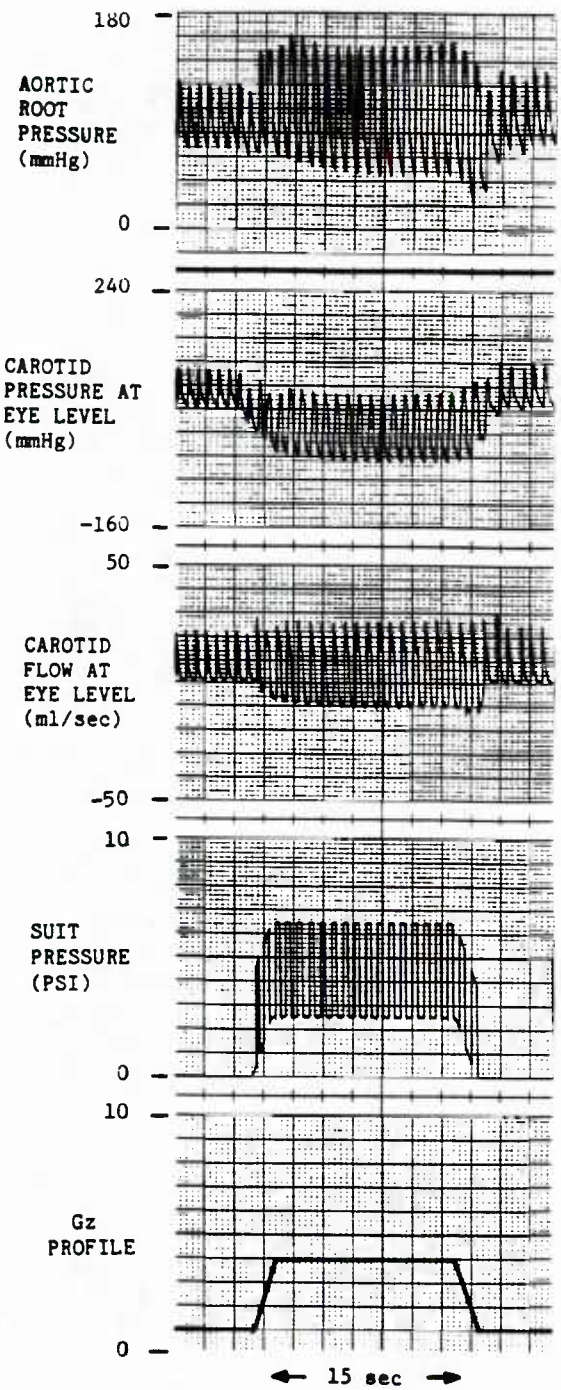

Figure 3 - Model output for subject wearing standard anti-G suit with external pulsation during cardiac systole superimposed on the suit pressure. See text for details.

Table 1: Model predictions for peripheral and central light loss.

mode	G_z value for beginning of peripheral light loss (PLL)	G_z value for central light loss (CLL)
unprotected	2.7	3.6
standard AGS	3.9	5.3
SEP (legs only)	3.2	4.0
abdominal AGS plus leg SEP	4.0	5.2
SEP superimposed on standard AGS	4.8	6.2

PRELIMINARY EXPERIMENTS

We carried out a limited series of human experiments to test the predictions of the model, and to investigate the practical limitations of applying SEP in a high G_z environment. Six male subjects, ages 21 to 43 who had previous experience in centrifuge experiments, participated in this study. Subjects were instrumented for measurements of uncalibrated blood flow velocity in the superficial temporal artery using a directional Doppler ultrasound flowmeter (LM Electronics model 1012), and uncalibrated blood volume changes in the index finger using an optical plethysmograph (Medical System Corporation, Great Neck, Long Island). In addition, the electrocardiogram, heart rate, G_z level, and the pressure in the lower portion of the anti-G suit were monitored. A closed circuit TV monitor provided continuous observation of the subject. Voice contact was maintained through a two way communication system.

The subjects wore the abdominal segment of a standard Naval anti-G suit which was inflated according to the standard AGS pressure regimen (inflation above +2.0 G_z with pressure set to 1.5 psi/G). Specially designed plastic bladders were applied around the thighs and calves of the subjects. The leg bladders were connected through a 3-way valve to a pressure source set to 8.5 psi. A relief valve set to 9 psi ensured that no higher pressure could be delivered to the bladders. Synchronization to the R-wave of the electrocardiogram was controlled by a microprocessor control unit developed by us and modified for these experiments [23]. The desired phasing of inflation and deflation was achieved by observing the plethysmograph waveform and manually adjusting the delay time between the R-wave and inflation. The bladders were alternately inflated to a pressure 8.5 psi and deflated to atmosphere in synchrony with the cardiac cycle. At these pressures, approximately 125 msec was required to fully inflate or deflate the bladders. Inflation of the bladders during various parts of the cardiac

cycle was tested.

Subjects were seated in the NADC (Naval Air Development Center, Warminster, PA) human centrifuge at a 15 degree seat back angle, and were subjected to a $+G_z$ profile having 4 second haversine rise and fall times and lasting for 15 seconds at its plateau. This acceleration profile was repeated after a 1 minute rest period at a plateau level 0.5 G_z greater than the previous run. Plateau levels were increased until the subject failed to complete the 15 second run without losing peripheral vision (60 degrees of peripheral vision remaining as measured by the NADC light bar [8]).

Typical results of the centrifuge runs are shown in Figures 4-6. Figure 4 shows a sample response of an unprotected subject experiencing an acceleration stress of +3 G_z. From top to bottom, Figure 4 depicts ECG, doppler flow velocity, heart rate (uncalibrated), G_z profile and the signal corresponding to the pilot's tracking of his peripheral vision. Note the recovery of temporal artery blood flow velocity which begins in the middle of the run. Figures 5 and 6 show samples of the responses during synchronized external pulsation in two different subjects experiencing +3.5 G_z. In these samples, the external pressure pulses were phased to augment systemic pressure during cardiac diastole. These runs were selected for presentation to demonstrate the effect of pumping on the measureable variables (when SEP was applied during systole, the change in the form of the waves was not as obvious). From top to bottom, the figures present doppler flow velocity, ECG, leg cuff pressure, plethysmograph signal, pump trigger signal, G_z profile, and abdominal segment suit pressure. Note the augmentation in the diastolic portion of the plethysmograph and the temporal blood flow signals when the external pulsation system was activated. Also note that the heart rate of the subject in Figure 6 was higher than that of Figure 5, and that diastolic augmentation in this subject as observed in the plethysmograph tracing was lower than that of the subject of Figure 5.

A comparison between tolerance levels measured in our experiments and those predicted by the model is shown in Table 2. For the purpose of this comparison, the driving parameters of the model were adjusted to correspond to the practical experimental conditions. Specifically, model leg cuff pressure was set to vary between the experimentally recorded positive maximum and atmosphere because our experimental leg bladders were vented to atmosphere. The rates of inflation and deflation were also adjusted to correspond to the slower experimental values. For all values involving SEP, the pressurized period coincided with cardiac systole. The first column of the table lists the average G_z value at which the subjects reported only 60 degrees of peripheral vision remaining. For the model's prediction (second column) we assumed that 60 degrees of peripheral vision remains at an eye level pressure of 30 mmHg. This value was obtained by interpolating between the pressure at which peripheral light loss begins (50 mmHg) and that at which central light loss is reached (20 mmHg). There is good agreement between model and experiment. Note, however, that the model predictions are slightly lower than the experimental data. Also note that a significant improvement in tolerance is predicted with increased rates of inflation and deflation of the bladders (third column).

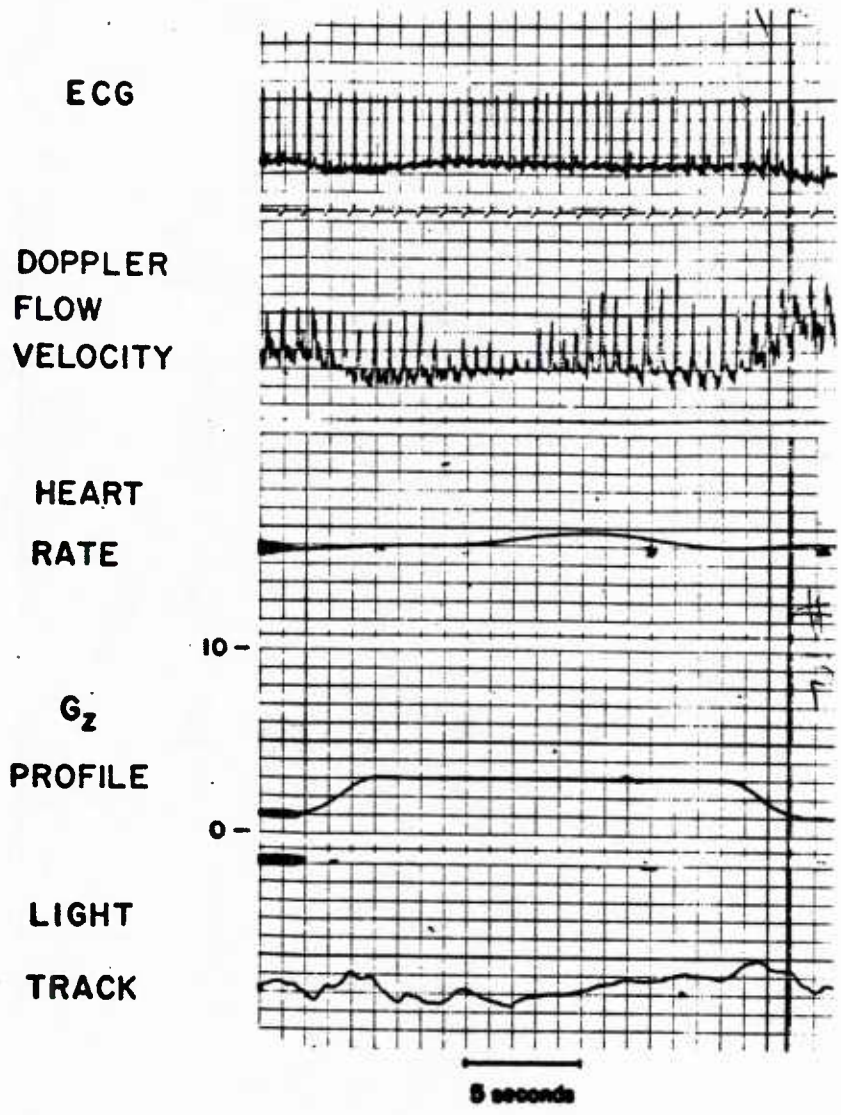

Figure 4 - Example of recordings during human centrifuge run of an unprotected subject.

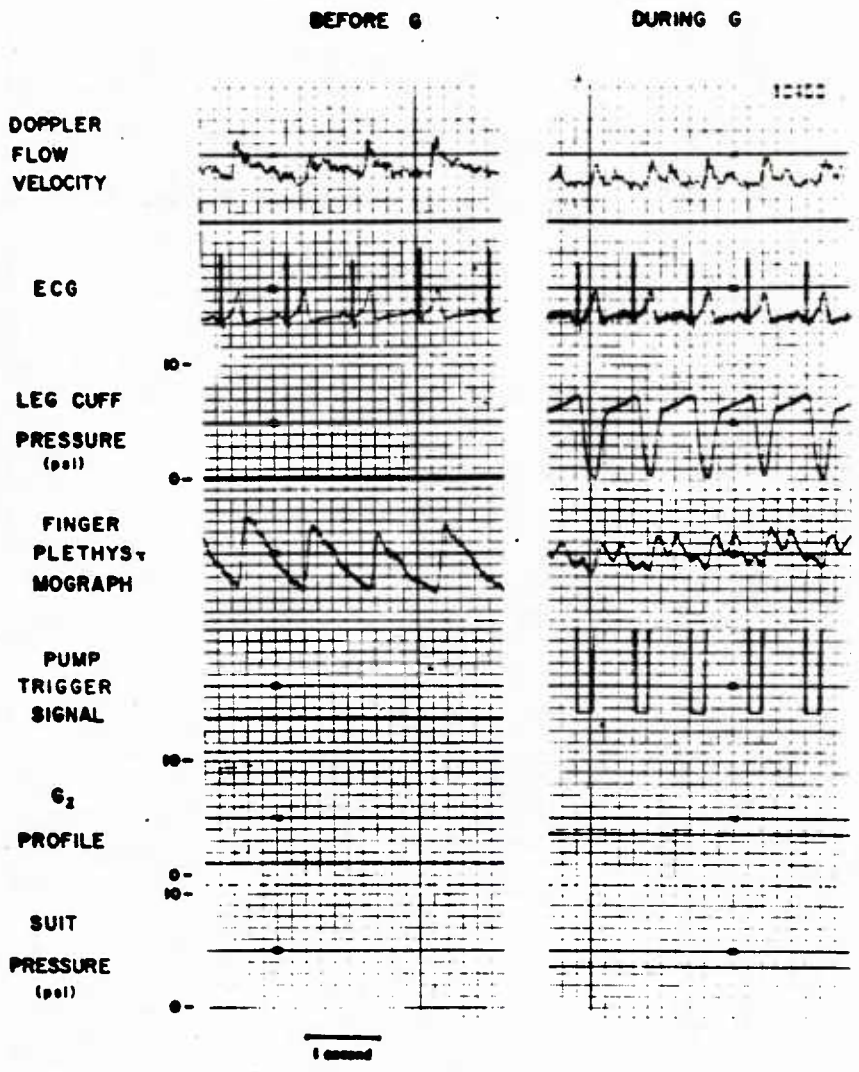

Figure 5 - Recordings during a centrifuge run of a subject wearing an abdominal segment of an AGS with external pulsation applied to the leg bladders. Leg bladders were pressurized during cardiac diastole.

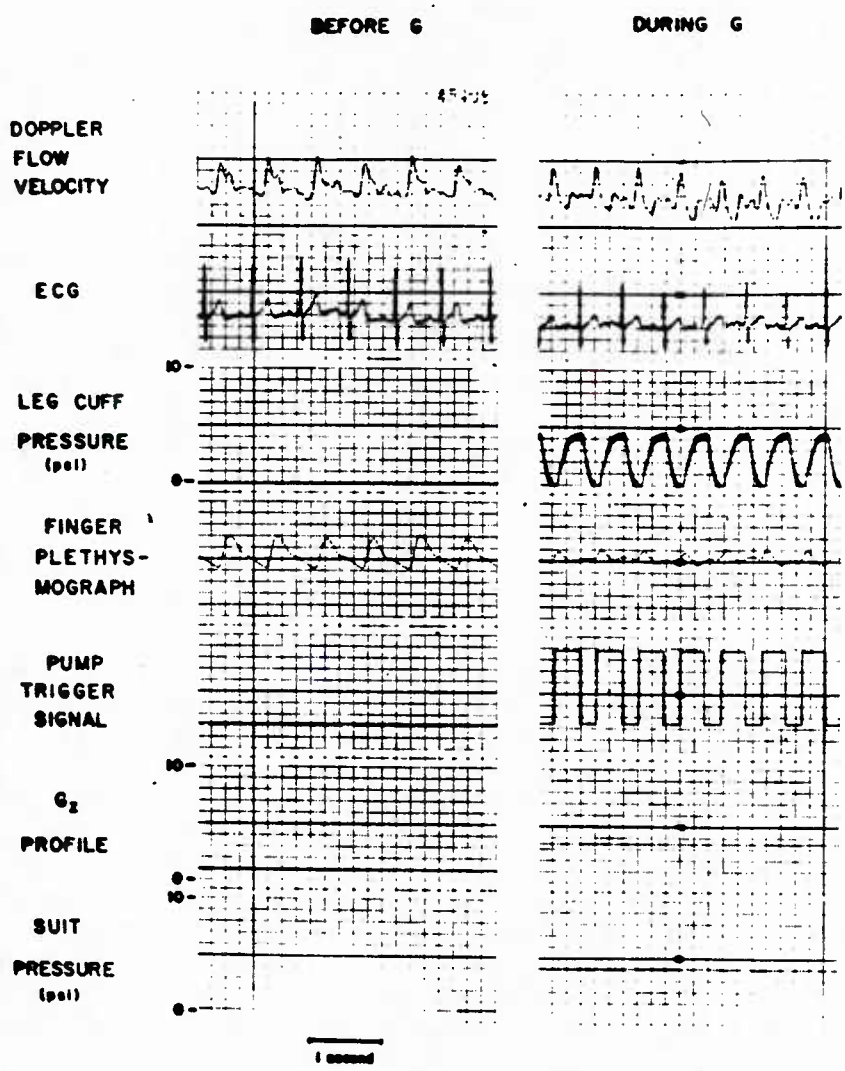

Figure 6 - Recordings during a centrifuge run of a subject wearing an abdominal segment of an AGS with external pulsation applied to the leg bladders. Leg bladders were pressurized during cardiac diastole.

Table 2. Comparison between predicted and measured G tolerance. (n = number of subjects)

	n	Gz tolerance* (experimental data)	Gz tolerance* (model simulation)	Gz tolerance* (model, fast rise time)
unprotected	2	3.5	3.3	
standard AGS	6	5.2**	4.8	
SEP (legs only)	5	3.6	3.4	3.7
abdominal AGS plus leg SEP	6	4.7	4.3	4.8

* G_z tolerance was defined as that acceleration at which only 60 degrees of peripheral vision remained. In the model, this was assumed to occur at an eye level pressure of 30 mmHg. See text for details.

** Based on unpublished data from experiments at NADC using same subjects.

DISCUSSION

Several important features of the behavior of the cardiovascular system under acceleration stress predicted by our model are in agreement with reports in the literature and with our own preliminary experimental observations. Those of interest to the present investigation include the values of protected and unprotected acceleration tolerance and the effects of the compensatory mechanisms of the cardiovascular system in response to pressure changes during G. Of the several modes of external pressure simulated by our model, systolic pulsation with a pressure of 2 psi superimposed on both the abdominal and leg segments of the standard AGS achieved the greatest improvement in tolerance to G_z stress. In the preliminary experimental runs, pulsation of all suit bladders was not possible. However, with partial suit pulsation, tolerance values predicted by the model were very close to those realized in the experiments.

For the unprotected subject, our model predicts that for a $+G_z$ profile of 1 G/sec rise time and 15 second duration, peripheral light loss (PLL) begins at +2.7 G_z, while central light loss (CLL) occurs at +3.6 G_z. To arrive at these numbers, we assumed that even a momentary episode of reduced pressure to CLL or PLL level was sufficient for vision impairment. The results of the model computations for the unprotected subject, shown in Figure 1, indicate an initial drop in ophthalmic artery pressure at G_z onset, followed by a partial recovery during the plateau stage. This recovery is due to the physiologic compensatory mechanisms incorporated into the model. Similar observations have previously been reported in human experiments [14,28], and have also been made by us from doppler velocity measurements during the centrifuge runs. An example is shown in Figure 4. Blood flow velocity in the superficial temporal artery, after an initial drop at the onset of maximum G, displays a partial recovery toward the end of the run. (Note that this signal is subject to baseline

drift, so that only peak to peak variations are significant).

In Figures 2 and 3, for the subject protected by the standard and pulsating G suit pressures, the model predicts a smaller initial drop in eye level pressure, and it does not indicate a recovery attributable to the compensatory mechanism during the plateau stage. These results can be explained on the basis of the pressure difference between the sites of the ophthalmic artery and that of the carotid sinus. The compensatory mechanism incorporated in our model operates on the basis of carotid sinus pressure which, in the presence of antegrade flow in the upright position, is always higher than the pressure at the ophthalmic artery. The magnitude of this difference increases with increased $+G_z$. Although the ophthalmic artery pressure under +4 G_z acceleration (in Figures 2 and 3) is lower than the pre-G value, our calculations indicate that the pressure at the carotid sinus level has indeed been returned close to the pre-G value by the action of the anti-G suit. Therefore, only small changes in eye level pressure due to physiologic compensation are to be expected. Nevertheless, model predictions suggest that a significant increase in mean blood flow to the brain is present. This augmentation in mean blood flow beyond that expected from the augmentation in pressure, is a result of the non-linear behavior of the peripheral vasculature. Specifically, as mean pressure approaches zero, the peripheral resistance undergoes large changes in value during each cardiac cycle as a function of the pulsatile pressure and the response of our postulated vessel collapse mechanism. Because of this mechanism, the level of increase in peripheral blood flow depends upon the extent (amplitude and time duration) by which the pulsatile pressure exceeds the collapse pressure of the peripheral vasculature during each heart beat. This augmentation in mean blood flow takes place at G levels close to the subject's tolerance limit and is more evident toward the latter part of the plateau phase of the G profile.

The decline in eye level pressure toward the end of the G_z plateau predicted by the model at high G_z levels has also been observed by Shubrooks in human experiments [28]. These results may explain in part the decrease in acceleration tolerance toward the end of experimental centrifuge runs observed by Shubrooks and by us.

The results obtained from the model (Table 1) suggest that synchronized external pulsation using 2 psi, superimposed on both the abdominal and leg segments of the standard AGS, is capable of augmenting tolerance to acceleration stress by 0.9 G above the protection afforded by the standard AGS alone. This figure is likely to be an underestimate of the actual increase in protection because the effects of external pressure on the venous system are not as yet incorporated into the model. The model indicates that pulsating only the leg bladders while inflating the abdominal segments to the standard regimen does not increase acceleration stress protection. If sufficiently high, the constant abdominal pressure may prevent the effects of the leg bladders from reaching the upper circulation.

The preliminary results obtained from our human centrifuge runs show good agreement with model predictions. The model's predictions of G_z tolerance (Table 2) are somewhat lower than those measured experimentally, probably due to the lack of coupling of the external pressure to the venous system in the model. The results also suggest that the technique of synchronized external pulsation is feasible in the high G environment,

although practical implementation of an optimal system has not been achieved. Augmentation of diastolic pressure by diastolic pressurization of the leg bladders is clearly evident on both the plethysmograph and doppler tracings of Figure 5. However, the inability of our system to inflate and deflate both the abdominal and leg bladders at the required rates prevented verification of the model's predictions of added G_z protection. Even with only the leg bladders pulsating, inflation was too slow for the fast heart rate of Figure 6. Diastolic augmentation was therefore reduced from that shown in Figure 5 where the subject's heart rate was slower. It is expected that this difficulty will be overcome in the future through the use of an improved pressure delivery system and specially designed bladders.

The investigation suggests two important conclusions relative to the use of synchronized external pulsation as a method for improved G tolerance:

1. The model predicts a potentially substantial improvement in tolerance with the use of synchronized external pulsation of all three suit segments as seen in Table 1.

2. Increasing the rates of inflation and deflation improves the results of synchronized external pulsation as seen in Table 2. This can be achieved by minimizing the volume of the anti-G suit.

The results of this investigation demonstrate that the predictions of our model compare very favorably with experimental observations, and that the model may be a useful tool in investigating the behavior of hemodynamic and physiological variables under G_z acceleration.

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